measured 0.5 cm. in diameter. The histological picture was that of calcification, with no repair.

Experiment 23. A rabbit, 1950 grams in weight, was injected on January 9. It was killed March 14; weight, 2700 grams. In the upper portion of the abdominal aorta was one small wrinkled thickening 0.3 mm. in diameter, which presents histologically the picture of calcification.

These results, while they offer no explanation of the mode of action of adrenalin in producing vascular lesions, simplify the mode of investigation, in that the problem is limited to the study of the effect of single injections, and comparisons may be drawn sharply between lesions so produced and similar lesions caused by the prolonged administration of other substances, such as nicotin, digalen, and barium chloride. It is evident, however, that in view of these results the theory of direct toxic action is hardly tenable, while that which recognizes a combination of vasovasorum disturbance, local anemia, and vascular strain is greatly strengthened.

BACILLUS PYROGENES (NOV. SPEC.?) ASSOCIATED WITH A FEBRILE DISEASE.

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The following account of an unusual disease with the pigmented bacillus isolated from it, is published on account of its general interest and the impossibility of identifying the organism associated with it. The descriptions of most pigmented bacteria are so meager and faulty, especially the absence of animal experiments, that it is dangerous at this day to announce a new species. I even hesitate to go so far as to conclude from the evidence that the bacillus is the etiological factor and responsible for the disease. Yet it seems from the tissue changes in inoculated animals that this bacillus produces a characteristic, well-definable pathological condition.

A search through the literature has yielded no evidence of an organism described completely enough to compare fully with Bacillus pyrogenes (nov. spec.?). This name has been selected at the suggestion of Professor William H. Welch, to whom I am much indebted.

On January 2, 1906, a gentleman thirty-six years of age, who had a slight fever, was sent from Lynchburg, Virginia, to Prof. George J. Preston, of Baltimore. The patient was taken to the Baltimore City Hospital, suffering with a number of nervous symptoms, and slight fever; his temperature ranged between 99° and 100° and 101°, and reached 102° F. only once. His most striking symptoms were meningeal: photophobia, hyperesthesia, restlessness, tremors, both coarse and fine; Kernig's sign was present, and various joints were swollen and painful at different times.

An examination of the patient's blood for typhoid bacilli was negative. A blood count showed a leukocytosis of about 14,000. The patient had a typhoid appearance, but had no rose spots or enlarged spleen. The most striking feature of his symptoms was the involvement of the joints; as one subsided another flared up. The patient refused permission to take cultures from the joints. Prof. Preston felt convinced, from the start, that the case was not one of infection from typhoid bacilli. On February 20, the patient still had fever, whereupon Prof. Preston kindly asked me to make a blood culture. The patient was finally discharged on March 15, 1906. He has remained perfectly well since.

During the patient's presence in the hospital it was possible to make one only agglutination test with the organism which was isolated from his blood. At the end of two hours no agglutination had taken place.

The three Ehrlenmeyer flasks into which the 10 c.c. of blood had been distributed were incubated at a temperature of 37° C., for seventy-two hours. Colonies grew from only one flask.

The surface colonies after twenty-four hours showed themselves on the average about the size of a pin-head. They were small, round, and lemon-colored, and varied in shape from a perfect circle to that of an oval; colonies were slightly raised, shiny, somewhat translucent, and moist. By an inverted No. 3 eye-piece they were yellow, finely granular, with more or less regular edges. The deeper colonies were more concentrated, smaller, and contained a darker nucleus.

On slant agar the growth remained confined to the line of the needle; the stroke was a very deep lemon-yellow color. It was diffuse, irregularly filiform, moist, slightly raised, and shiny.

After seventy-two hours the growth seemed to have reached its maximum. At this time, compared to a seventy-two-hour-old typhoid culture, it was about twice its thickness and twice its width.

Cover-glass preparations contained a small diplobacillus, with rounded ends and almost as broad as long. It measured from 0.6 to 1.4 μ long by 0.5 to 0.8 μ thick. It was completely discolorized by Gram's stain. No spores were found. Capsules were present. It was a flagellated organism of the peritrichal variety.

Bouillon had faint cloudiness. Loops of bouillon in hanging-drop preparations showed very actively motile bacilli in pairs and short chains. The motility was equal to or exceeded that of the typhoid bacillus.

In gelatin the puncture gave a very characteristic growth after twenty-four hours. On the surface for a few days the growth was no larger than the width of the needle. After the fifth day the surface growth spread to a millimeter in diameter, and took on a deep-yellow color. In the depth of the gelatin, along the stab, the growth spread very slightly, became somewhat granular and filamentous, with a pale-yellow tint. In all of the gelatin stabs a beautiful spiral of discrete granular colonies began to appear at the bottom of the stab about the fourth day. Until the seventh day there was no sign of liquefaction, after which it liquefied rapidly.

The growth on potatoes was thick, opaque, and profuse; dark lemon-yellow in color, raised, with a velvety surface, shiny and dry.

Litmus milk became very slightly acid at the end of twenty-four hours. At the end of two weeks many small particles of coagulated casein were seen floating on the surface of the milk.

On peptone there was a cloudy, yellow growth.

No indol was formed.

Fermentation tubes, which contain lactose bouillon, glucose bouillon, or saccharose bouillon, when inoculated with Bacillus pyrogenes, produced no gas.

Litmus agar, lactose litmus agar, and mannite litmus agar were acidulated.

The thermal death-point of Bacillus pyrogenes, exposed for ten minutes, is 60°.

There was no odor, pleasant or unpleasant, attached to any of the cultures.

The pathogenic power of Bacillus pyrogenes was tested by inoculating four rabbits and two guinea-pigs intravenously, intraperitoneally, and subcutaneously. The two former methods, when employed in these experiments, were very effective in killing the animals. Animals inoculated subcutaneously did not die.

Four cubic centimeters of a twenty-four-hour bouillon culture of Bacillus pyrogenes was inoculated into the ear vein of a large gray rabbit at 11 A.M. February 27, 1906. It was alive and feeding, but not very active, at 3.30 P.M. At 4 P.M. it was found dead. The autopsy was performed at 12.30 P.M. February 28.

A second rabbit was inoculated with 4 c.c. of a twenty-four-hour old pure bouillon culture of Bacillus pyrogenes on February 27. Inoculation was made into the left hind knee or second joint. On February 28 a small amount of fluid seemed to be present and some capitus was felt; this remained until March 7, when the rabbit was found dead.

A third rabbit (small albino) was inoculated in the ear vein on March 1, at 2 P.M., with 0.5 c.c. of a pure bouillon culture of Bacillus pyrogenes. The rabbit was killed on March 4. Pure cultures of the organism originally inoculated were obtained from the blood and all the viscera. A description of this autopsy tallies exactly with autopsy No. 1.

A large male guinea-pig inoculated in the peritoneal cavity with 1 c.c. of a twenty-four-hour bouillon culture of Bacillus pyrogenes on April 7, at 1 P.M.; the guinea-pig was found dead.

Pure cultures of Bacillus pyrogenes were obtained from all these autopsies. A complete description of them and of the tissues is reported in another place.

After searching the literature carefully I have been unable to find any organism which in the slightest degree resembles this one. Dr. Stokes called my attention to the description of an organism found in Manila, P. I., and reported by Lieutenant Maximilian Herzog. After carefully comparing these organisms, his and the one I report, I cannot find the slightest resemblance. His organism was obtained at an autopsy performed by him on an unknown native islander who was brought to the hospital dead. The patient's previous history was entirely unknown. At the autopsy the only lesions found were general glandular enlargements and an incised wound in the left leg. The liver, spleen, and kidney showed parenchymatous degeneration. The liver showed some interstitial hepatitis.

Cultures taken from the various organs, after twenty-four hours, showed no growth except that taken from the liver. The culture made from the liver grew an organism with a heavy golden-yellow pigment and exuding a fœtid cadaverous odor; hence, Dr. Herzog has called his organism (ignoring the binominal nomenclature) "Bacillus aureus fœtidus."

It will be easier, perhaps, to compare his organism with the one isolated by me from Prof. Preston's patient, by placing them side by side in the following table:

	Bacillus pyrogenes.	*Bacillus aureus fœtidus.*
Pigment	Lemon yellow	Golden yellow
Capsule	Absent	Present
Spores	Negative	Positive
Motility	Active	Absent
Size	0.6 micron to 2.1 microns	0.4 micron to 1.4 microns long
	0.5 micron to 0.8 micron	0.4 micron to 1.4 microns wide
Liquefaction	Complete in twelve hours	Absent until seventh day
Odor	None	Cadaverous
Death-point	60°	62°

To summarize, a male patient, aged thirty-six years, with a temperature lasting eight weeks, which never rose above 102°, exhibited meningeal symptoms, photophobia, tremors, irritability, subsultus tendinum, and joint involvement; the Widal reaction was constantly absent; there was a leukocytosis of 14,000. Cultures taken from his blood revealed a lemon-yellow diplobacillus pathogenic for guinea-pigs and rabbits, negative to Gram's stain, actively motile, producing no indol, not liquefying gelatin until the seventh day, and producing no gas in lactose, saccharose, or glucose media. Milk, mannite, litmus agar were acidified. The organism was killed at an exposure of 60° C. for ten minutes. It has been named Bacillus pyrogenes.

In conclusion, I wish to extend thanks for kind suggestions and assistance to Prof. George J. Preston, through whose generosity I was enabled to do this work; to Prof. Wm. H. Welch, for naming the organism, and especially to Prof. Wm. Royal Stokes, for much assistance.

CERTAIN REMOTE CONSEQUENCES OF INFECTIONS OF THE BILIARY TRACT, WITH SPECIAL REFERENCE TO, (1) CHOLELITHIASIS AND CHOLECYSTITIS, (2) ADHESIONS OF THE UPPER ABDOMEN, (3) THE GENERAL PRINCIPLES OF TREATMENT, AND (4) THE INDICATIONS FOR SURGICAL INTERVENTION.[1]

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In discussing the more immediate consequences of infection of the biliary tract,[2] I mentioned that virulent bacteria and definite anatomical lesions may persist in the gall-bladder for years, and that such subacute or chronic, insidious, and long-standing infections play a major role in the etiology of gallstones, cholecystitis, pericholecystic adhesions, and the divers symptoms commonly but often erroneously ascribed to disorder of the stomach.

THE INFECTIOUS NATURE OF CHOLELITHIASIS, CHOLECYSTITIS, AND THEIR COMPLICATIONS.

In asking your attention now to a discussion of the infectious nature of gallstones and the entire cholelithitic process, it is not my intention to review in detail the manner of formation of gallstones; this was done some years ago by the master hand of Naunyn. But I may point out briefly the accepted opinions on the subject.

There is general agreement that the one necessary factor in the

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[1] Part II of the Mütter Lecture of the College of Physicians of Philadelphia, delivered December 1, 1905. Part I of the Lecture, entitled "Infections of the Biliary Tract, with Special Reference to Latent (or Masked) and Typhoid Infections," was published in the Amer. Jour. Med. Sci., September, 1906, cxxxii, 446.

[2] Part I of this Lecture, loc. cit.